STIC Search Report
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STIC Database Tracking Number: 95428

To: Joseph F Murphy **Location:** CM1-10D19 **Art Unit:** 1646 Tuesday, June 03, 2003 **Case Serial Number:** 09/672020	**From:** Beverly Shears **Location:** Biotech-Chem Library CM1-1E05 **Phone:** 308-4994 beverly.shears@uspto.gov

Search Notes

Murphy
09/672020

FILE 'REGISTRY' ENTERED AT 15:46:57 ON 03 JUN 2003
L1 54 S AVAEIQLMH[3.]K/SQSP

FILE 'HCAPLUS' ENTERED AT 15:47:43 ON 03 JUN 2003
L2 13 S L1

L2 ANSWER 1 OF 13 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2003:84125 HCAPLUS
DOCUMENT NUMBER: 138:281271
TITLE: Functional Evidence for an Intramolecular Side Chain Interaction between Residues 6 and 10 of Receptor-Bound Parathyroid Hormone Analogues
AUTHOR(S): Shimizu, Naoto; Petroni, Brian D.; Khatri, Ashok; Gardella, Thomas J.
CORPORATE SOURCE: Endocrine Unit, Massachusetts General Hospital, Boston, MA, 02114, USA
SOURCE: Biochemistry (2003), 42(8), 2282-2290
CODEN: BICHAW; ISSN: 0006-2960
PUBLISHER: American Chemical Society
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The N-terminal domain of PTH(1-34) is crit. for PTH-1 receptor (P1R) activation and has been postulated to be .alpha.-helical when bound to the receptor. The authors investigated the possibility that the side chains of residues 6 (Gln) and 10 (Gln or Asn) of PTH analogs, which would align on the same face of the predicted .alpha.-helix, could interact and thereby contribute to the PTH/P1R interaction process. The authors utilized PTH(1-11), PTH(1-14), and PTH(1-34) analogs substituted with alanine at one or both of these positions and functionally evaluated the peptides in cell lines (HKRK-B7 and HKRK-B28) stably expressing the P1R, as well as in COS-7 cells transiently expressing either the P1R or a P1R construct that lacks the N-terminal extracellular domain (P1R-DelNt). In HKRK-B7 cells, the single substitutions of Gln6 .fwdarw. Ala and Gln10 .fwdarw. Ala reduced the cAMP-stimulating potency of [Ala3,Gln10,Arg11]rPTH(1-11)NH2 .apprx.60- and .apprx.2-fold, resp., whereas the combined Ala6,10 substitution resulted in a .apprx.2-fold gain in potency, relative to the single Ala6 substitution. Similar effects on P1R-mediated cAMP-signaling potency and P1R-binding affinity were obsd. for these substitutions in [Aib1,3,Gln10,Har11,Ala12,Trp14]rPTH(1-14)NH2. Installation of a lactam bridge between the Lys6 and the Glu10 side chains of [Ala3,12,Lys6,Glu10,Har11,Trp14]rPTH(1-14)NH2 increased signaling potency 6-fold, relative to the nonbridged linear analog. Alanine substitutions at positions 6 and/or 10 of [Tyr34]hPTH(1-34)NH2 did not affect signaling potency nor binding affinity on the intact P1R; however, Ala6 abolished PTH(1-34) signaling on P1R-DelNt, and this effect was reversed by Ala10. The overall data support the hypothesis that the N-terminal portion of PTH is .alpha.-helical when bound to the activation domain of the PTH-1 receptor and they further suggest that intrahelical side chain interactions between residues 6 and 10 of the ligand can contribute to the receptor interaction process.

IT **357417-44-8**
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(functional evidence for intramol. side chain interaction of receptor-bound parathyroid hormone analogs)

REFERENCE COUNT: 35 THERE ARE 35 CITED REFERENCES AVAILABLE

FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 2 OF 13 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:760607 HCAPLUS
DOCUMENT NUMBER: 138:33479
TITLE: Residue 19 of the parathyroid hormone (PTH) modulates ligand interaction with the juxtamembrane region of the PTH-1 receptor
AUTHOR(S): Shimizu, Masaru; Shimizu, Naoto; Tsang, Janet C.; Petroni, Brian D.; Khatri, Ashok; Potts, John T., Jr.; Gardella, Thomas J.
CORPORATE SOURCE: Endocrine Unit, Massachusetts General Hospital and Harvard Medical School, Boston, MA, 02114, USA
SOURCE: Biochemistry (2002), 41(44), 13224-13233
CODEN: BICHAW; ISSN: 0006-2960
PUBLISHER: American Chemical Society
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Recent data suggest that the binding of parathyroid hormone (PTH)-(1-34) to the PTH-1 receptor (P1R) involves a high-affinity interaction between the C-terminal (15-34) domain of the ligand and the N-terminal extracellular (N) domain of the receptor and a low-affinity interaction between the N-terminal (1-14) portion of PTH and the juxtamembrane (J) region of the receptor, with the latter interaction giving rise to signal transduction. The authors investigated whether residues C-terminal of position 14 in PTH(1-34) contribute to the J component of the interaction mechanism by comparing the capacity of PTH analogs N-terminally modified to improve J domain affinity and C-terminally truncated at position 14, 20, or 34 to stimulate cAMP formation in COS-7 cells transiently transfected with P1R-delNt, a P1R construct that lacks most of the N domain. In these cells, the potency of [M]PTH(1-34) (M = Ala1,3,12,Gln10,Har11,Trp14,Arg19) was 120-fold greater than that of [M]PTH(1-14) (EC50s = 3.0 and 360 nM, resp.) but was equal to that of [M]PTH(1-20) (EC50 = 2.3 nM). Reverting the Arg19 substitution of [M]PTH(1-20) to the native Glu reduced cAMP signaling potency on P1R-delNt by 12-fold (EC50 of [M]PTH(1-20)-Glu19 = 27 nM), and it decreased the analog's capacity to inhibit the binding of the J domain-selective radioligand, 125I-[Aib1,3,Nle8,M,Tyr21]ratPTH(1-21), to the full-length P1R stably expressed in LLC-PK1 cells by 40-fold. The Glu19 .fwdarw. Arg modification, however, did not affect the capacity of PTH(15-31) to inhibit the binding of the N domain-selective radioligand 125I-bPTH(3-34) to the full-length receptor. The overall data suggest that residues (15-20) of PTH, and particularly residue 19, contribute to the capacity of the N-terminal portion of the ligand to interact with the juxtamembrane region of the receptor. The NMR data presented in the accompanying manuscript suggests that this role could involve intramol. effects on secondary structure in the N-terminal portion of the ligand.

IT **293299-19-1 477946-15-9 477946-16-0 477946-17-1 477946-18-2 477951-24-9 478897-24-4 478897-25-5**
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(parathyroid hormone modified analogs binding by juxtamembrane region of PTH-1 receptor and signal induction in relation to

structure)
REFERENCE COUNT: 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 3 OF 13 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:760606 HCAPLUS
DOCUMENT NUMBER: 138:419
TITLE: Residue 19 of the Parathyroid Hormone: Structural Consequences
AUTHOR(S): Piserchio, Andrea; Shimizu, Naoto; Gardella, Thomas J.; Mierke, Dale F.
CORPORATE SOURCE: Department of Chemistry, Division of Biology & Medicine, Brown University, Providence, RI, 02912, USA
SOURCE: Biochemistry (2002), 41(44), 13217-13223
CODEN: BICHAW; ISSN: 0006-2960
PUBLISHER: American Chemical Society
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Residue 19 of the parathyroid hormone (PTH) has been shown to play an important role in both binding to and activation of the PTH receptor; specifically, Arg19-contg. analogs have improved biol. function over similar Glu19 peptides. Addnl. the juxtamembrane portion of the receptor is involved in the different biol. responses. Here, the authors det. the conformational preferences of PTH analogs to provide a structural basis for their biol. actions. On the basis of CD results, the Arg19 .fwdarw. Glu19 mutations within the context of both PTH(1-20) and PTH(1-34) analogs lead to increases in helix content, ranging from a 8-15% increase. High-resoln. structures as detd. by 1H NMR and NOE-restrained mol. dynamics simulations clearly illustrate the difference between Arg19 and Glu19-PTH(1-20), particularly with the extent and stability of the C-terminal helix. The Arg19-contg. analog has a well defined, stable .alpha.-helix from Ser4-Arg19, while the Glu19 analog is less ordered at the C-terminus. On the basis of these observations, the authors propose that position 19 of PTH(1-20) must be .alpha.-helical for optimal interaction with the juxtamembrane portion of the receptor. This mode of binding extends the current view of PTH binding (indeed ligand binding for all class B GPCRs), which invokes a bihelical ligand with the C-terminus of the ligand interacting with the N-terminus of the receptor (responsible for binding) and the N-terminus of the ligand interacting with the seven-helical bundle (leading to receptor activation).
IT **476683-18-8 476683-20-2**
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(parathyroid hormone structure in relation to receptor binding)
REFERENCE COUNT: 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 4 OF 13 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:692637 HCAPLUS
DOCUMENT NUMBER: 138:131299
TITLE: Minimization of parathyroid hormone using simultaneous multiple peptide synthesis: implications for structure based drug design

AUTHOR(S): Khatri, Ashok; Huang, Xiang-Chen; Petroni, Brian D.; Gardella, Thomas J.
CORPORATE SOURCE: Massachusetts General Hospital, Boston, MA, 02114, USA
SOURCE: Peptides: The Wave of the Future, Proceedings of the Second International and the Seventeenth American Peptide Symposium, San Diego, CA, United States, June 9-14, 2001 (2001), 890-891. Editor(s): Lebl, Michal; Houghten, Richard A. American Peptide Society: San Diego, Calif. CODEN: 69DBAL; ISBN: 0-9715560-0-8
DOCUMENT TYPE: Conference
LANGUAGE: English

AB Using a multiple peptide synthesizer, a series of parathyroid hormone (PTH) (1-14) analogs was synthesized and used to define the structure-activity relationships in the peptide and potentially improve potency. The 1-9 region of PTH was relatively intolerant to substitution, while 10-14 region was tolerant. Activity-enhancing effect are possible with substitutions at positions 3, 10, 11, 12, and 14. The enhancing effects are additive as [M]PTH(1-14) is 1000-fold more potent than PTH. The 10-14 region is amenable to protein engineering, and side-chain aromaticity, polarizability and length are beneficial at position 11.

IT **357417-44-8P**
RL: BSU (Biological study, unclassified); PRP (Properties); SPN (Synthetic preparation); BIOL (Biological study); PREP (Preparation)
(minimization of parathyroid hormone using simultaneous multiple peptide synthesis in relation to implications for structure based drug design)

REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 5 OF 13 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:43337 HCAPLUS
DOCUMENT NUMBER: 136:241809
TITLE: Parathyroid hormone (PTH)-(1-14) and -(1-11) analogs conformationally constrained by .alpha.-aminoisobutyric acid mediate full agonist responses via the juxtamembrane region of the PTH-1 receptor
AUTHOR(S): Shimizu, Naoto; Guo, Jun; Gardella, Thomas J.
CORPORATE SOURCE: Endocrine Unit, Massachusetts General Hospital and Harvard Medical School, Boston, MA, 02114, USA
SOURCE: Journal of Biological Chemistry (2001), 276(52), 49003-49012
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The N-terminal portion of parathyroid hormone is crit. for PTH-1 receptor (P1R) activation and has been postulated to be .alpha.-helical when bound to the receptor. We investigated whether substitution of the sterically hindered and helix-promoting amino acid .alpha.-aminoisobutyric acid (Aib) in N-terminal PTH oligopeptides would improve the capacity of the peptide to activate

the P1R. Anal. of the effects of individual Aib substitutions at each position in [Ala3,12,Gln10,Har11,Trp14]PTH(1-14)NH2 ([M]PTH(1-14)) on cAMP-stimulating potency in HKRK-B28 cells revealed that Aib at most positions diminished potency; however, Aib at positions 1 and 3 enhanced potency. Thus [Aib1,3,M]PTH(1-14) was .apprx.100-fold more potent than [M]PTH(1-14) (EC50 = 1.1 and 100 nM, resp.), .apprx.100,000-fold more potent than native PTH(1-14), and 2-fold more potent than PTH(1-34). The shorter peptide, [Aib1,3,M]PTH(1-11), was also fully efficacious and 1000-fold more potent than [M]PTH(1-11) (EC50 4 nM vs. 3 .mu.M). In cAMP stimulation assays performed in COS-7 cells expressing P1R-delNt, a receptor that lacks most of the N-terminal extracellular domain, [Aib1,3,M]PTH(1-14) was 50-fold more potent than [M]PTH(1-14) (EC50 = 0.7 vs. 40 nM) and 1000-fold more potent than PTH(1-34) (EC50 = 700 nM). [Aib1,3,M]PTH(1-14), but not PTH(1-34), inhibited the binding of 125I-[Aib1,3,Nle8,Gln10,Har11,Ala12,Trp14,Arg19,Tyr21]PTH (1-21)NH2 to hP1R-delNt (IC50 = 1600 nM). The Aib1,3 substitutions in otherwise unmodified PTH(1-34) enhanced potency and binding affinity on hP1R-delNt, but they had no effect for this peptide on hP1R-WT. CD spectroscopy demonstrated that the Aib-1,3 substitutions increased helicity in all peptides tested, including PTH(1-34). The overall data thus suggest that the N-terminal residues of PTH are intrinsically disordered but become conformationally constrained, possibly as an .alpha.-helix, upon interaction with the activation domain of the PTH-1 receptor.

IT **357417-44-8 403990-60-3 403990-61-4**
403990-63-6 403990-65-8
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(PTH-(1-14) and -(1-11) analogs conformationally constrained by aminoisobutyric acid mediate full agonist responses via juxtamembrane region of PTH-1 receptor)

REFERENCE COUNT: 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 6 OF 13 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:694423 HCAPLUS
DOCUMENT NUMBER: 135:353089
TITLE: Parathyroid hormone receptor internalization is independent of protein kinase A and phospholipase C activation
AUTHOR(S): Tawfeek, Hesham A. W.; Che, Jian; Qian, Fang; Abou-Samra, Abdul B.
CORPORATE SOURCE: Endocrine Unit, Massachusetts General Hospital and Harvard Medical School, Boston, MA, 02114, USA
SOURCE: American Journal of Physiology (2001), 281(3, Pt. 1), E545-E557
CODEN: AJPHAP; ISSN: 0002-9513
PUBLISHER: American Physiological Society
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Parathyroid hormone (PTH) and PTH-related peptide (PTHrP) binding to their common receptor stimulates second messenger accumulation, receptor phosphorylation, and internalization. LLC-PK1 cells expressing a green fluorescent protein-tagged PTH/PTHrP receptor show time- and dose-dependent receptor internalization. The internalized receptors colocalize with clathrin-coated pits.

Internalization is stimulated by PTH analogs that bind to and activate the PTH/PTHrP receptor. Cell lines expressing a mutant protein kinase A regulatory subunit that is resistant to cAMP and/or a mutant receptor (DSEL mutant) that does not activate phospholipase C internalize their receptors normally. In addn., internalization of the wild-type receptor and the DSEL mutant is stimulated by the PTH analog [Gly1,Arg19]hPTH-(1-28), which does not stimulate phospholipase C. Forskolin, IBMX, and the active phorbol ester, phorbol-12-myristate-13-acetate, did not promote receptor internalization or increase PTH-induced internalization. These data indicate that ligand-induced internalization of the PTH/PTHrP receptor requires both ligand binding and receptor activation but does not involve stimulation of adenylate cyclase/protein kinase A or phospholipase C/protein kinase C.

IT **332139-36-3 332139-40-9 372957-00-1**
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(parathyroid hormone receptor internalization is independent of adenylate/protein kinase A and phospholipase C/protein kinase C activation as characterized by PTH analogs in COS-7 and LLC-PK1 cells)

REFERENCE COUNT: 76 THERE ARE 76 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 7 OF 13 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:473648 HCAPLUS
DOCUMENT NUMBER: 135:205634
TITLE: Enhanced activity in parathyroid hormone-(1-14) and -(1-11): novel peptides for probing ligand-receptor interactions
AUTHOR(S): Shimizu, Masaru; Carter, Percy H.; Khatri, Ashok; Potts, John T., Jr.; Gardella, Thomas J.
CORPORATE SOURCE: Endocrine Unit, Massachusetts General Hospital and Harvard Medical School, Boston, MA, 02114, USA
SOURCE: Endocrinology (2001), 142(7), 3068-3074
CODEN: ENDOAO; ISSN: 0013-7227
PUBLISHER: Endocrine Society
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The N-terminal portion of PTH is crit. for PTH-1 receptor (P1Rc) activation. In exploring this component of the ligand receptor interaction, the authors recently showed that the agonist potency of the weakly active PTH-(1-14)NH2 peptide can be enhanced by natural amino acid substitutions at several positions, including position 11 (normally leucine). Here the potency of PTH-(1-14)NH2 can be enhanced by using nonnatural amino acids that increase the length and polarizability of the position 11 side-chain. Thus, in LLC-PK1 cells stably expressing high levels of the human P1Rc, [homoarginine(Har)11]PTH-(1-14)NH2 was 30-fold more potent for cAMP prodn. than was native PTH-(1-14)NH2. Combining the homoarginine-11 substitution with other recently identified activity-enhancing substitutions yielded [Ala3,12,Gln10,Har11,Trp11]PTH-(1-14)NH2, which was 1500-fold more potent than PTH-(1-14)NH2 (EC50 = 0.12 and 190 .mu.M, resp.) and only 63-fold less potent than PTH-(1-34) (EC50 = 1.9 nM). The even shorter analog [Ala3,Gln10,Har11]PTH-(1-11)NH2

was also a full cAMP agonist (EC50 = 3.1 .mu.M). Receptor mutations at Phe184 and Leu187 located near the boundary of the N-terminal domain and transmembrane domain-1 severely impaired responsiveness to the PTH-(1-11) analog. Overall, these studies demonstrate that PTH analogs of only 11 amino acids are sufficient for activation of the PTH-1 receptor through interaction with its juxtamembrane region.

IT **293299-20-4 293299-21-5 357417-43-7 357417-44-8**
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(parathyroid hormone fragment analogs signaling activity in relation to structure)

REFERENCE COUNT: 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 8 OF 13 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:293253 HCAPLUS
DOCUMENT NUMBER: 135:56189
TITLE: Zinc(II)-mediated enhancement of the agonist activity of histidine-substituted parathyroid hormone(1-14) analogues
AUTHOR(S): Carter, P. H.; Gardella, T. J.
CORPORATE SOURCE: Endocrine Unit, Massachusetts General Hospital and Harvard Medical School, Boston, MA, 02114, USA
SOURCE: Biochimica et Biophysica Acta (2001), 1538(2-3), 290-304
CODEN: BBACAQ; ISSN: 0006-3002
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Previous studies on parathyroid hormone (PTH)(1-14) revealed that residues (1-9) played a dominant role in stimulating PTH-1 receptor-mediated increases in cAMP formation. In the present study, we examd. the effects of installing a metal-binding motif in the (10-14) region of rat PTH(1-14) on the peptide's agonist activity. We found that substitution of histidine for the native asparagine at position 10 of PTH(1-14) provided a peptide that was approx. 8-fold more potent as an agonist in the presence of divalent zinc salts than it was in the absence of the metal. This enhancement in potency was dependent on the native histidine at position 14, the concn. of Zn(II) utilized, and did not occur with other divalent metal ions. The zinc-activated [His10]-PTH(1-14) peptide was blocked by a classical PTH-1 receptor antagonist, PTHrP(7-36), and did not activate the PTH-2 receptor. The zinc-mediated enhancing effect did not require the large N-terminal extracellular domain of the PTH-1 receptor. Although we were able to demonstrate that [His10]-PTH(1-14) binds Zn(II) using 1H-NMR, our spectroscopic studies (CD and NMR) were not consistent with the notion that zinc enhanced the activity of [His10]-PTH(1-14) simply by inducing a helical structure in the 10-14 region. Rather, the data suggest that the enhancement in cAMP potency arises from the formation of a ternary complex between [His10]-PTH(1-14), a zinc atom, and the extracellular loop/transmembrane domain region of the PTH-1 receptor.

IT **345643-09-6**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(zinc(II)-mediated enhancement of agonist activity of histidine-substituted parathyroid hormone(1-14) analogs)
REFERENCE COUNT: 41 THERE ARE 41 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 9 OF 13 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:247456 HCAPLUS
DOCUMENT NUMBER: 134:276166
TITLE: Preparation of polypeptide derivatives of parathyroid hormone (PTH) and their use in diagnosis and therapy of bone resorption disorders
INVENTOR(S): Gardella, Thomas J.; Kronenberg, Henry M.; Potts, John T., Jr.; Juppner, Harald
PATENT ASSIGNEE(S): The General Hospital Corporation, USA
SOURCE: PCT Int. Appl., 97 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001023521	A2	20010405	WO 2000-US26818	20000929
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 2000077348	A5	20010430	AU 2000-77348	20000929

PRIORITY APPLN. INFO.:
US 1999-156927P P 19990929
US 2000-185060P P 20000225
WO 2000-US26818 W 20000929

OTHER SOURCE(S): MARPAT 134:276166

AB Novel parathyroid hormone (PTH) polypeptide derivs. are disclosed, as are pharmaceutical compns. contg. said polypeptides, and synthetic and recombinant methods for producing said polypeptides. Also disclosed are methods for treating mammalian conditions characterized by decreases in bone mass using therapeutically effective pharmaceutical compns. contg. said polypeptides. Also disclosed are methods for screening candidate compds. of the invention for antagonistic or agonistic effects on parathyroid hormone receptor action. Also disclosed are diagnostic and therapeutic methods of said compds.

IT **293299-18-0P 293299-19-1P 293299-20-4P**
293299-21-5P 293299-25-9P 332139-36-3P
332139-39-6P 332139-40-9P 332139-41-0P

332139-42-1P 332345-97-8P 332345-98-9P
332346-54-0P 332346-55-1P 333318-24-4P
333318-25-5P 333318-26-6P 333330-89-5P
RL: BAC (Biological activity or effector, except adverse); BPN (Biosynthetic preparation); BSU (Biological study, unclassified); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(prepn. of polypeptide derivs. of parathyroid hormone and use in diagnosis and therapy of bone resorption disorders)

L2 ANSWER 10 OF 13 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:247375 HCAPLUS
DOCUMENT NUMBER: 134:276165
TITLE: Polypeptide derivatives of parathyroid hormone for the treatment of bone and cartilage disorders
INVENTOR(S): Gardella, Thomas J.; Kronenberg, Henry M.; Potts, John T.; Jueppner, Harald
PATENT ASSIGNEE(S): The General Hospital Corporation, USA
SOURCE: PCT Int. Appl., 100 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001023427	A1	20010405	WO 2000-US4716	20000225
W: CA, JP				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
EP 1222208	A1	20020717	EP 2000-910323	20000225
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY				
JP 2003511014	T2	20030325	JP 2001-526577	20000225

PRIORITY APPLN. INFO.: US 1999-156927P P 19990929
WO 2000-US4716 W 20000225

OTHER SOURCE(S): MARPAT 134:276165

AB Novel parathyroid hormone (PTH) polypeptide derivs. are disclosed, as are pharmaceutical compns. contg. said polypeptides, and synthetic and recombinant methods for producing said polypeptides. Also disclosed are methods for treating mammalian conditions characterized by decreases in bone mass using therapeutically effective pharmaceutical compns. contg. said polypeptides. Also disclosed are methods for screening candidate compds. of the invention for antagonistic or agonistic effects on parathyroid hormone receptor action. Also disclosed are diagnostic and therapeutic methods of said compds.

IT **332139-36-3P 332139-39-6P 332139-40-9P**
332139-41-0P 332139-42-1P 332345-97-8P
332345-98-9P 332346-54-0P 332346-55-1P
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(polypeptide derivs. of parathyroid hormone for the treatment of bone and cartilage disorders)

IT **333403-48-8 333403-52-4 333403-57-9**
333403-68-2 333403-71-7 333403-73-9
333403-75-1 333403-78-4 333403-82-0
333403-88-6
RL: PRP (Properties)
(unclaimed protein sequence; polypeptide derivs. of parathyroid hormone for the treatment of bone and cartilage disorders)
REFERENCE COUNT: 16 THERE ARE 16 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 11 OF 13 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:216266 HCAPLUS
DOCUMENT NUMBER: 134:305466
TITLE: Evaluating the signal transduction mechanism of the parathyroid hormone 1 receptor. Effect of receptor-G-protein interaction on the ligand binding mechanism and receptor conformation
AUTHOR(S): Hoare, Sam R. J.; Gardella, Thomas J.; Usdin, Ted B.
CORPORATE SOURCE: Unit on Cell Biology, Laboratory of Genetics, National Institute of Mental Health, Bethesda, MD, 20892-4092, USA
SOURCE: Journal of Biological Chemistry (2001), 276(11), 7741-7753
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Ligand binding to the PTH1 receptor is described by a "two-site" model, in which the C-terminal portion of the ligand interacts with the N-terminal domain of the receptor (N interaction), and the N-terminal region of the ligand binds the juxtamembrane domain of the receptor (J interaction). Previous studies have not considered the dynamic nature of receptor conformation in ligand binding and receptor activation. In this study the ligand binding mechanism was compared for the G-protein-coupled (RG) and uncoupled (R) PTH1 receptor conformations. The two-site model was confirmed by demonstration of spatially distinct binding sites for PTH(3-34) and PTH(1-14): PTH(1-14), which binds predominantly to the J domain, only partially inhibited binding of 125I-PTH(3-34); and PTH(3-34), shown to bind predominantly to the N domain, only partially inhibited PTH(1-14)-stimulated cAMP accumulation. To assess the effect of R-G coupling, ligand binding to R was measured by displacement of 125I-PTH(3-34) with 30 .mu.M guanosine 5'-3-O-(thio)triphosphate (GTP.gamma.S) present, and binding to RG was measured by displacement of 125I-[MAP]PTHrP(1-36) (where MAP is model amphipathic peptide), a new radioligand that binds selectively to RG. Agonists bound with higher affinity to RG than R, whereas antagonists bound similarly to these states. The J interaction was responsible for enhanced agonist binding to RG: residues 1 and 2 were required for increased PTH(1-34) affinity for RG; residue 5 of MAP-PTHrP(1-36) was a determinant of R/RG binding selectivity, and PTH(1-14) bound selectively to RG. The N interaction was insensitive to R-G coupling; PTH(3-34) binding was GTP.gamma.S-insensitive. Finally, several observations suggest the receptor conformation is more "closed" at RG than R. At the R

state, an open conformation is suggested by the simultaneous binding of PTH(1-14) and PTH(3-34). At RG PTH(1-14) better occluded binding of 125I-PTH(3-34) and agonist ligands bound pseudo-irreversibly, suggesting a more closed conformation of this receptor state. The results extend the two-site model to take into account R and RG conformations and suggest a model for differences of receptor conformation between these states.

IT **293299-19-1 335242-13-2,**
[Ala1,3,10,12.Arg11,19]hPTH(1-34)
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(structural detn. of parathyroid hormone 1 receptor ligand binding and signaling and receptor-G-protein interaction effect on ligand binding mechanism and receptor conformation)

REFERENCE COUNT: 57 THERE ARE 57 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 12 OF 13 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2000:515055 HCAPLUS
DOCUMENT NUMBER: 133:247378
TITLE: Minimization of parathyroid hormone. Novel amino-terminal parathyroid hormone fragments with enhanced potency in activating the type-1 parathyroid hormone receptor
AUTHOR(S): Shimizu, Masaru; Potts, John T., Jr.; Gardella, Thomas J.
CORPORATE SOURCE: Endocrine Unit, Massachusetts General Hospital and Harvard Medical School, Boston, MA, 02114, USA
SOURCE: Journal of Biological Chemistry (2000), 275(29), 21836-21843
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The amino-terminal and carboxyl-terminal portions of the 1-34 fragment of parathyroid hormone (PTH) contain the major determinants of receptor activation and receptor binding, resp. We investigated how the amino-terminal signaling portion of PTH interacts with the receptor by utilizing analogs of the weakly active fragment, rat (r) PTH(1-14)NH2, and cells transfected with the wild-type human PTH-1 receptor (hP1R-WT) or a truncated PTH-1 receptor which lacked most of the amino-terminal extracellular domain (hP1R-delNt). Of 132 mono-substituted PTH(1-14) analogs, most having substitutions in the (1-9) region were inactive in assays of cAMP formation in LLC-PK1 cells stably expressing hP1R-WT, whereas most having substitutions in the (10-14) region were active. Several substitutions (e.g. Ser3 .fwdarw. Ala, Asn10 .fwdarw. Ala or Gln, Leu11 .fwdarw. Arg, Gly12 .fwdarw. Ala, His14 .fwdarw. Trp) enhanced activity 2-10-fold. These effects were additive, as [Ala3,10,12,Arg11,Trp14] rPTH(1-14)NH2 was 220-fold more potent than rPTH(1-14)NH2 (EC50 = 0.6 and 133 .mu.M, resp.). Native rPTH(1-11) was inactive, but [Ala3,10,Arg11]rPTH(1-11)NH2 achieved maximal cAMP stimulation (EC50 = 17 .mu.M). The modified PTH fragments induced cAMP formation with hP1R-delNt in COS-7 cells as potently as they did with hP1R-WT;

PTH(1-34) was 6,000-fold weaker with hP1R-delNt than with hP1R-WT. The most potent analog, [Ala3,10,12,Arg11,Trp14]rPTH(1-14)NH2, stimulated inositol phosphate prodn. with hP1R-WT. The results show that short NH2-terminal peptides of PTH can be optimized for considerable gains in signaling potency through modification of interactions involving the regions of the receptor contg. the transmembrane domains and extracellular loops.

IT **293299-05-5 293299-09-9 293299-10-2**
293299-11-3 293299-15-7 293299-16-8
293299-18-0 293299-19-1 293299-20-4
293299-21-5 293299-25-9 294199-44-3
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(parathyroid hormone N-terminal fragments with enhanced potency in activating type-1 parathyroid hormone receptor)

REFERENCE COUNT: 44 THERE ARE 44 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 13 OF 13 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1999:288751 HCAPLUS
DOCUMENT NUMBER: 131:83087
TITLE: The (1-14) fragment of parathyroid hormone (PTH) activates intact and amino-terminally truncated PTH-1 receptors
AUTHOR(S): Luck, Michael D.; Carter, Percy H.; Gardella, Thomas J.
CORPORATE SOURCE: Endocrine Unit, Massachusetts General Hospital and Harvard Medical School, Boston, MA, 02114, USA
SOURCE: Molecular Endocrinology (1999), 13(5), 670-680
CODEN: MOENEN; ISSN: 0888-8809
PUBLISHER: Endocrine Society
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Recent mutagenesis and crosslinking studies suggest that residues in the carboxyl-terminal portion of PTH(1-34) interact with the amino-terminal extracellular domain of the receptor and thereby contribute strongly to binding energy; and that residues in the amino-terminal portion of the ligand interact with the receptor region contg. the transmembrane helixes and extracellular loops and thereby induce second messenger signaling. We investigated the latter component of this hypothesis using the short amino-terminal fragment PTH(1-14) and a truncated rat PTH-1 receptor (r.DELTA.Nt) that lacks most of the amino-terminal extracellular domain. The binding of PTH(1-14) to LLC-PK1 or COS-7 cells transfected with the intact PTH-1 receptor was too weak to detect; however, PTH(1-14) dose-dependently stimulated cAMP formation in these cells over the dose range of 1-100 .mu.M. PTH(1-14) also stimulated cAMP formation in COS-7 cells transiently transfected with r.DELTA.Nt, and its potency with this receptor was nearly equal to that seen with the intact receptor. In contrast, PTH(1-34) was .apprx.100-fold weaker in potency with r.DELTA.Nt than it was with the intact receptor. Alanine scanning of PTH(1-14) revealed that for both the intact and truncated receptors, the 1-9 segment of PTH forms a crit. receptor activation domain. Taken together, these results demonstrate that the amino-terminal portion of PTH(1-34) interacts with the

juxtamembrane regions of the PTH-1 receptor and that these interactions are sufficient for initiating signal transduction.

IT **229616-37-9**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(parathyroid hormone N-terminal fragment activation of intact and N-terminally truncated PTH-1 receptors)

REFERENCE COUNT: 55 THERE ARE 55 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

E50 THROUGH E103 ASSIGNED

FILE 'REGISTRY' ENTERED AT 15:49:04 ON 03 JUN 2003

L3 54 SEA FILE=REGISTRY ABB=ON PLU=ON (293299-19-1/BI OR 357417-44-8/BI OR 293299-20-4/BI OR 293299-21-5/BI OR 332139-36-3/BI OR 332139-40-9/BI OR 293299-18-0/BI OR 293299-25-9/BI OR 332139-39-6/BI OR 332139-41-0/BI OR 332139-42-1/BI OR 332345-97-8/BI OR 332345-98-9/BI OR 332346-54-0/BI OR 332346-55-1/BI OR 229616-37-9/BI OR 293299-05-5/BI OR 293299-09-9/BI OR 293299-10-2/BI OR 293299-11-3/BI OR 293299-15-7/BI OR 293299-16-8/BI OR 294199-44-3/BI OR 333318-24-4/BI OR 333318-25-5/BI OR 333318-26-6/BI OR 333330-89-5/BI OR 333403-48-8/BI OR 333403-52-4/BI OR 333403-57-9/BI OR 333403-68-2/BI OR 333403-71-7/BI OR 333403-73-9/BI OR 333403-75-1/BI OR 333403-78-4/BI OR 333403-82-0/BI OR 333403-88-6/BI OR 335242-13-2/BI OR 345643-09-6/BI OR 357417-43-7/BI OR 372957-00-1/BI OR 403990-60-3/BI OR 403990-61-4/BI OR 403990-63-6/BI OR 403990-65-8/BI OR 476683-18-8/BI OR 476683-20-2/BI OR 477946-15-9/BI OR 477946-16-0/BI OR 477946-17-1/BI OR 477946-18-2/BI OR 477951-24-9/BI OR 478897-24-4/BI OR 478897-25-5/BI)

L4 54 L1 AND L3

L4 ANSWER 1 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **478897-25-5** REGISTRY
CN L-Tyrosine, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-alanyl-L-arginyl-L-alanyl-L-lysyl-L-histidyl-L-leucyl-L-asparaginyl-L-seryl-L-methionyl-L-.alpha.-glutamyl-L-arginyl-L-valyl-L-.alpha.-glutamyl-L-tryptophyl-L-leucyl-L-arginyl-L-lysyl-L-lysyl-L-leucyl-L-glutaminyl-L-.alpha.-aspartyl-L-valyl-L-histidyl-L-asparaginyl- (9CI) (CA INDEX NAME)
CI MAN
SQL 34

SEQ 1 AVAEIQLMHA RAKHLNSMER VEWLRKKLQD VHNY
========== ===
HITS AT: 1-13

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 138:33479

L4 ANSWER 2 OF 54 REGISTRY COPYRIGHT 2003 ACS

RN **478897-24-4** REGISTRY
CN L-Tyrosinamide, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-alanyl-L-arginyl-L-alanyl-L-lysyl-L-histidyl-L-leucyl-L-asparaginyl-L-seryl-L-methionyl-L-arginyl-L-arginyl-L-valyl-L-.alpha.-glutamyl-L-tryptophyl-L-leucyl-L-arginyl-L-lysyl-L-lysyl-L-leucyl-L-glutaminyl-L-.alpha.-aspartyl-L-valyl-L-histidyl-L-asparaginyl- (9CI) (CA INDEX NAME)
CI MAN
SQL 34

SEQ 1 AVAEIQLMHA RAKHLNSMRR VEWLRKKLQD VHNY
========== ===
HITS AT: 1-13

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 138:33479

L4 ANSWER 3 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **477951-24-9** REGISTRY
CN L-Tyrosinamide, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-asparaginyl-N6-(aminoiminomethyl)-L-lysyl-L-alanyl-L-lysyl-L-tryptophyl-L-leucyl-L-asparaginyl-L-seryl-L-methionyl-L-arginyl-L-arginyl-L-valyl-L-.alpha.-glutamyl-L-tryptophyl-L-leucyl-L-arginyl-L-lysyl-L-lysyl-L-leucyl-L-glutaminyl-L-.alpha.-aspartyl-L-valyl-L-histidyl-L-asparaginyl- (9CI) (CA INDEX NAME)
CI MAN
SQL 34

SEQ 1 AVAEIQLMHN KAKWLNSMRR VEWLRKKLQD VHNY
========== ===
HITS AT: 1-13

REFERENCE 1: 138:33479

L4 ANSWER 4 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **477946-18-2** REGISTRY
CN L-Tryptophanamide, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-asparaginyl-N6-(aminoiminomethyl)-L-lysyl-L-alanyl-L-lysyl- (9CI) (CA INDEX NAME)
SQL 14

SEQ 1 AVAEIQLMHN XAKW
========== ===
HITS AT: 1-13

REFERENCE 1: 138:33479

L4 ANSWER 5 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **477946-17-1** REGISTRY
CN L-Argininamide, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-asparaginyl-N6-(aminoiminomethyl)-L-lysyl-L-alanyl-L-lysyl-L-tryptophyl-L-leucyl-L-asparaginyl-L-seryl-L-methionyl-L-.alpha.-glutamyl- (9CI) (CA INDEX NAME)

SQL 20

SEQ 1 AVAEIQLMHN XAKWLNSMER
========== ===
HITS AT: 1-13

REFERENCE 1: 138:33479

L4 ANSWER 6 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **477946-16-0** REGISTRY
CN L-Argininamide, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-asparaginyl-N6-(aminoiminomethyl)-L-lysyl-L-alanyl-L-lysyl-L-tryptophyl-L-leucyl-L-asparaginyl-L-seryl-L-methionyl-L-arginyl- (9CI) (CA INDEX NAME)
SQL 20

SEQ 1 AVAEIQLMHN XAKWLNSMRR
========== ===
HITS AT: 1-13

REFERENCE 1: 138:33479

L4 ANSWER 7 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **477946-15-9** REGISTRY
CN L-Argininamide, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-alanyl-L-arginyl-L-alanyl-L-lysyl-L-histidyl-L-leucyl-L-asparaginyl-L-seryl-L-methionyl-L-arginyl- (9CI) (CA INDEX NAME)
SQL 20

SEQ 1 AVAEIQLMHA RAKHLNSMRR
========== ===
HITS AT: 1-13

REFERENCE 1: 138:33479

L4 ANSWER 8 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **476683-20-2** REGISTRY
CN L-Arginine, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-glutaminyl-N6-(aminoiminomethyl)-L-lysyl-L-alanyl-L-lysyl-L-tryptophyl-L-leucyl-L-asparaginyl-L-seryl-L-methionyl-L-.alpha.-glutamyl- (9CI) (CA INDEX NAME)
SQL 20

SEQ 1 AVAEIQLMHQ XAKWLNSMER
========== ===
HITS AT: 1-13

REFERENCE 1: 138:419

L4 ANSWER 9 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **476683-18-8** REGISTRY
CN L-Arginine, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-glutaminyl-N6-(aminoiminomethyl)-L-lysyl-L-alanyl-L-lysyl-L-tryptophyl-L-leucyl-L-asparaginyl-L-seryl-L-methionyl-L-arginyl- (9CI) (CA INDEX NAME)
SQL 20

SEQ 1 AVAEIQLMHQ XAKWLNSMRR
========== ===
HITS AT: 1-13

REFERENCE 1: 138:419

L4 ANSWER 10 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **403990-65-8** REGISTRY
CN Alaninamide, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-glutaminyl-N6-(aminoiminomethyl)-L-lysyl-L-alanyl-L-lysyl-2-methyl- (9CI) (CA INDEX NAME)
SQL 14

SEQ 1 AVAEIQLMHQ XAKX
========== ===
HITS AT: 1-13

REFERENCE 1: 136:241809

L4 ANSWER 11 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **403990-63-6** REGISTRY
CN L-Tryptophanamide, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-glutaminyl-N6-(aminoiminomethyl)-L-lysyl-2-methylalanyl-L-lysyl- (9CI) (CA INDEX NAME)
SQL 14

SEQ 1 AVAEIQLMHQ XXKW
========== ===
HITS AT: 1-13

REFERENCE 1: 136:241809

L4 ANSWER 12 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **403990-61-4** REGISTRY
CN L-Tryptophanamide, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-glutaminyl-2-methylalanyl-L-alanyl-L-lysyl- (9CI) (CA INDEX NAME)
SQL 14

SEQ 1 AVAEIQLMHQ XAKW
========== ===
HITS AT: 1-13

REFERENCE 1: 136:241809

L4 ANSWER 13 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **403990-60-3** REGISTRY
CN L-Tryptophanamide, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-2-methylalanyl-N6-(aminoiminomethyl)-L-lysyl-L-alanyl-L-lysyl- (9CI) (CA INDEX NAME)
SQL 14

SEQ 1 AVAEIQLMHX XAKW
========== ===

HITS AT: 1-13

REFERENCE 1: 136:241809

L4 ANSWER 14 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **372957-00-1** REGISTRY
CN L-Tryptophan, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-glutaminyl-N6-(aminoiminomethyl)-L-lysyl-L-alanyl-L-lysyl- (9CI) (CA INDEX NAME)
SQL 14

SEQ 1 AVAEIQLMHQ XAKW
========== ===
HITS AT: 1-13

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 135:353089

L4 ANSWER 15 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **357417-44-8** REGISTRY
CN L-Tryptophanamide, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-glutaminyl-N6-(aminoiminomethyl)-L-lysyl-L-alanyl-L-lysyl- (9CI) (CA INDEX NAME)
SQL 14

SEQ 1 AVAEIQLMHQ XAKW
========== ===
HITS AT: 1-13

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 138:281271

REFERENCE 2: 138:131299

REFERENCE 3: 136:241809

REFERENCE 4: 135:205634

L4 ANSWER 16 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **357417-43-7** REGISTRY
CN L-Histidinamide, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-glutaminyl-N6-(aminoiminomethyl)-L-lysyl-L-alanyl-L-lysyl- (9CI) (CA INDEX NAME)
SQL 14

SEQ 1 AVAEIQLMHQ XAKH
========== ===
HITS AT: 1-13

REFERENCE 1: 135:205634

L4 ANSWER 17 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **345643-09-6** REGISTRY

CN L-Histidinamide, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-histidyl-L-leucylglycyl-L-lysyl- (9CI) (CA INDEX NAME)
SQL 14

SEQ 1 AVAEIQLMHH LGKH
========== ===
HITS AT: 1-13

REFERENCE 1: 135:56189

L4 ANSWER 18 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **335242-13-2** REGISTRY
CN L-Tyrosine, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-alanyl-L-arginyl-L-alanyl-L-lysyl-L-histidyl-L-leucyl-L-asparaginyl-L-seryl-L-methionyl-L-arginyl-L-arginyl-L-valyl-L-.alpha.-glutamyl-L-tryptophyl-L-leucyl-L-arginyl-L-lysyl-L-lysyl-L-leucyl-L-glutaminyl-L-.alpha.-aspartyl-L-valyl-L-histidyl-L-asparaginyl- (9CI) (CA INDEX NAME)
CI MAN
SQL 34

SEQ 1 AVAEIQLMHA RAKHLNSMRR VEWLRKKLQD VHNY
========== ===
HITS AT: 1-13

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 134:305466

L4 ANSWER 19 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **333403-88-6** REGISTRY
CN 9: PN: WO0123427 SEQID: 31 unclaimed protein (9CI) (CA INDEX NAME)
CI MAN
SQL 32

SEQ 1 AVAEIQLMHX XXKXLNSMXR VEWLRKKLQD VH
========== ===
HITS AT: 1-13

REFERENCE 1: 134:276165

L4 ANSWER 20 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **333403-82-0** REGISTRY
CN 8: PN: WO0123427 SEQID: 30 unclaimed protein (9CI) (CA INDEX NAME)
CI MAN
SQL 30

SEQ 1 AVAEIQLMHX XXKXLNSMXR VEWLRKKLQD
========== ===
HITS AT: 1-13

REFERENCE 1: 134:276165

L4 ANSWER 21 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **333403-78-4** REGISTRY
CN 7: PN: WO0123427 SEQID: 29 unclaimed protein (9CI) (CA INDEX NAME)
CI MAN

SQL 28

SEQ 1 AVAEIQLMHX XXKXLNSMXR VEWLRKKL
========== ===
HITS AT: 1-13

REFERENCE 1: 134:276165

L4 ANSWER 22 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **333403-75-1** REGISTRY
CN 6: PN: WO0123427 SEQID: 28 unclaimed protein (9CI) (CA INDEX NAME)
CI MAN
SQL 26

SEQ 1 AVAEIQLMHX XXKXLNSMXR VEWLRK
========== ===
HITS AT: 1-13

REFERENCE 1: 134:276165

L4 ANSWER 23 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **333403-73-9** REGISTRY
CN 5: PN: WO0123427 SEQID: 27 unclaimed protein (9CI) (CA INDEX NAME)
CI MAN
SQL 24

SEQ 1 AVAEIQLMHX XXKXLNSMXR VEWL
========== ===
HITS AT: 1-13

REFERENCE 1: 134:276165

L4 ANSWER 24 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **333403-71-7** REGISTRY
CN 4: PN: WO0123427 SEQID: 26 unclaimed protein (9CI) (CA INDEX NAME)
CI MAN
SQL 22

SEQ 1 AVAEIQLMHX XXKXLNSMXR VE
========== ===
HITS AT: 1-13

REFERENCE 1: 134:276165

L4 ANSWER 25 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **333403-68-2** REGISTRY
CN 3: PN: WO0123427 SEQID: 25 unclaimed protein (9CI) (CA INDEX NAME)
CI MAN
SQL 20

SEQ 1 AVAEIQLMHX XXKXLNSMXR
========== ===
HITS AT: 1-13

REFERENCE 1: 134:276165

L4 ANSWER 26 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **333403-57-9** REGISTRY

CN 1: PN: WO0123427 SEQID: 16 unclaimed protein (9CI) (CA INDEX NAME)
CI MAN
SQL 34

SEQ 1 AVAEIQLMHX XXKXLNSMER VEWLRKKLQD VHDX
========== ===
HITS AT: 1-13

REFERENCE 1: 134:276165

L4 ANSWER 27 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **333403-52-4** REGISTRY
CN 2: PN: WO0123427 SEQID: 2 unclaimed protein (9CI) (CA INDEX NAME)
CI MAN
SQL 14

SEQ 1 AVAEIQLMHX RAKX
========== ===
HITS AT: 1-13

REFERENCE 1: 134:276165

L4 ANSWER 28 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **333403-48-8** REGISTRY
CN 1: PN: WO0123427 SEQID: 1 unclaimed protein (9CI) (CA INDEX NAME)
CI MAN
SQL 14

SEQ 1 AVAEIQLMHX XXKX
========== ===
HITS AT: 1-13

REFERENCE 1: 134:276165

L4 ANSWER 29 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **333330-89-5** REGISTRY
CN L-Tyrosinamide, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-alanyl-L-arginyl-L-alanyl-L-lysyl-L-histidyl-L-leucyl-L-asparaginyl-L-seryl-L-methionyl-L-.alpha.-glutamyl-L-arginyl-L-valyl-L-.alpha.-glutamyl-L-tryptophyl-L-leucyl-L-arginyl-L-lysyl-L-lysyl-L-leucyl-L-glutaminyl-L-.alpha.-aspartyl-L-valyl-L-histidyl-L-.alpha.-aspartyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 31: PN: WO0123521 SEQID: 12 claimed protein
CI MAN
SQL 34

SEQ 1 AVAEIQLMHA RAKHLNSMER VEWLRKKLQD VHDY
========== ===
HITS AT: 1-13

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 134:276166

L4 ANSWER 30 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **333318-26-6** REGISTRY

CN L-Tyrosinamide, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-alanyl-L-arginyl-L-alanyl-L-lysyl-L-histidyl-L-leucyl-L-asparaginyl-L-seryl-L-methionyl-L-arginyl-L-arginyl-L-valyl-L-.alpha.-glutamyl-L-tryptophyl-L-leucyl-L-arginyl-L-lysyl-L-lysyl-L-leucyl-L-glutaminyl-L-.alpha.-aspartyl-L-valyl-L-histidyl-L-.alpha.-aspartyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 38: PN: WO0123521 SEQID: 24 claimed protein
CI MAN
SQL 34

SEQ 1 AVAEIQLMHA RAKHLNSMRR VEWLRKKLQD VHDY
========== ===
HITS AT: 1-13

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 134:276166

L4 ANSWER 31 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **333318-25-5** REGISTRY
CN L-Tyrosinamide, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-alanyl-L-arginyl-L-alanyl-L-lysyl-L-histidyl-L-leucyl-L-alanyl-L-seryl-L-valyl-L-arginyl-L-arginyl-L-methionyl-L-glutaminyl-L-tryptophyl-L-leucyl-L-arginyl-L-lysyl-L-lysyl-L-leucyl-L-glutaminyl-L-.alpha.-aspartyl-L-valyl-L-histidyl-L-.alpha.-aspartyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 36: PN: WO0123521 SEQID: 23 claimed protein
CI MAN
SQL 34

SEQ 1 AVAEIQLMHA RAKHLASVRR MQWLRKKLQD VHDY
========== ===
HITS AT: 1-13

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 134:276166

L4 ANSWER 32 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **333318-24-4** REGISTRY
CN L-Tyrosinamide, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-alanyl-L-arginyl-L-alanyl-L-lysyl-L-histidyl-L-leucyl-L-alanyl-L-seryl-L-valyl-L-.alpha.-glutamyl-L-arginyl-L-methionyl-L-glutaminyl-L-tryptophyl-L-leucyl-L-arginyl-L-lysyl-L-lysyl-L-leucyl-L-glutaminyl-L-.alpha.-aspartyl-L-valyl-L-histidyl-L-.alpha.-aspartyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 33: PN: WO0123521 SEQID: 20 claimed protein
CI MAN
SQL 34

SEQ 1 AVAEIQLMHA RAKHLASVER MQWLRKKLQD VHDY
========== ===

HITS AT: 1-13

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 134:276166

L4 ANSWER 33 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **332346-55-1** REGISTRY
CN L-Tyrosine, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-alanyl-L-arginyl-L-alanyl-L-lysyl-L-histidyl-L-leucyl-L-alanyl-L-seryl-L-valyl-L-.alpha.-glutamyl-L-arginyl-L-methionyl-L-glutaminyl-L-tryptophyl-L-leucyl-L-arginyl-L-lysyl-L-lysyl-L-leucyl-L-glutaminyl-L-.alpha.-aspartyl-L-valyl-L-histidyl-L-.alpha.-aspartyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 14: PN: WO0123427 SEQID: 20 claimed protein
CN 16: PN: WO0123521 SEQID: 20 claimed protein
CI MAN
SQL 34

SEQ 1 AVAEIQLMHA RAKHLASVER MQWLRKKLQD VHDY
========== ===
HITS AT: 1-13

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 134:276166

REFERENCE 2: 134:276165

L4 ANSWER 34 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **332346-54-0** REGISTRY
CN L-Tyrosine, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-alanyl-L-arginyl-L-alanyl-L-lysyl-L-histidyl-L-leucyl-L-asparaginyl-L-seryl-L-methionyl-L-.alpha.-glutamyl-L-arginyl-L-valyl-L-.alpha.-glutamyl-L-tryptophyl-L-leucyl-L-arginyl-L-lysyl-L-lysyl-L-leucyl-L-glutaminyl-L-.alpha.-aspartyl-L-valyl-L-histidyl-L-.alpha.-aspartyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 10: PN: WO0123521 SEQID: 12 claimed protein
CN 12: PN: WO0123427 SEQID: 12 claimed protein
CI MAN
SQL 34

SEQ 1 AVAEIQLMHA RAKHLNSMER VEWLRKKLQD VHDY
========== ===
HITS AT: 1-13

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 134:276166

REFERENCE 2: 134:276165

L4 ANSWER 35 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **332345-98-9** REGISTRY

CN L-Tyrosine, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-alanyl-L-arginyl-L-alanyl-L-lysyl-L-histidyl-L-leucyl-L-asparaginyl-L-seryl-L-methionyl-L-arginyl-L-arginyl-L-valyl-L-.alpha.-glutamyl-L-tryptophyl-L-leucyl-L-arginyl-L-lysyl-L-lysyl-L-leucyl-L-glutaminyl-L-.alpha.-aspartyl-L-valyl-L-histidyl-L-.alpha.-aspartyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 16: PN: WO0123427 SEQID: 24 claimed protein
CN 19: PN: WO0123521 SEQID: 24 claimed protein
CI MAN
SQL 34

SEQ 1 AVAEIQLMHA RAKHLNSMRR VEWLRKKLQD VHDY
========== ===
HITS AT: 1-13

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 134:276166

REFERENCE 2: 134:276165

L4 ANSWER 36 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **332345-97-8** REGISTRY
CN L-Tyrosine, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-alanyl-L-arginyl-L-alanyl-L-lysyl-L-histidyl-L-leucyl-L-alanyl-L-seryl-L-valyl-L-arginyl-L-arginyl-L-methionyl-L-glutaminyl-L-tryptophyl-L-leucyl-L-arginyl-L-lysyl-L-lysyl-L-leucyl-L-glutaminyl-L-.alpha.-aspartyl-L-valyl-L-histidyl-L-.alpha.-aspartyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 15: PN: WO0123427 SEQID: 23 claimed protein
CN 18: PN: WO0123521 SEQID: 23 claimed protein
CI MAN
SQL 34

SEQ 1 AVAEIQLMHA RAKHLASVRR MQWLRKKLQD VHDY
========== ===
HITS AT: 1-13

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 134:276166

REFERENCE 2: 134:276165

L4 ANSWER 37 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **332139-42-1** REGISTRY
CN L-Lysine, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-alanyl-L-arginyl-L-alanyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 7: PN: WO0123521 SEQID: 9 claimed sequence
CN 9: PN: WO0123427 SEQID: 9 claimed protein
SQL 13

SEQ 1 AVAEIQLMHA RAK
========== ===

HITS AT: 1-13

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 134:276166

REFERENCE 2: 134:276165

L4 ANSWER 38 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **332139-41-0** REGISTRY
CN L-Histidine, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-glutaminyl-L-arginyl-L-alanyl-L-lysyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 6: PN: WO0123521 SEQID: 8 claimed sequence
CN 8: PN: WO0123427 SEQID: 8 claimed protein
SQL 14

SEQ 1 AVAEIQLMHQ RAKH
========== ===
HITS AT: 1-13

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 134:276166

REFERENCE 2: 134:276165

L4 ANSWER 39 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **332139-40-9** REGISTRY
CN L-Tryptophan, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-alanyl-L-arginyl-L-alanyl-L-lysyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 5: PN: WO0123521 SEQID: 7 claimed sequence
CN 7: PN: WO0123427 SEQID: 7 claimed protein
SQL 14

SEQ 1 AVAEIQLMHA RAKW
========== ===
HITS AT: 1-13

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 135:353089

REFERENCE 2: 134:276166

REFERENCE 3: 134:276165

L4 ANSWER 40 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **332139-39-6** REGISTRY
CN L-Histidine, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-asparaginyl-L-arginyl-L-alanyl-L-lysyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 4: PN: WO0123521 SEQID: 6 claimed sequence
CN 6: PN: WO0123427 SEQID: 6 claimed protein

SQL 14

SEQ 1 AVAEIQLMHN RAKH
========== ===
HITS AT: 1-13

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 134:276166

REFERENCE 2: 134:276165

L4 ANSWER 41 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **332139-36-3** REGISTRY
CN L-Histidine, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-alanyl-L-arginyl-L-alanyl-L-lysyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 1: PN: WO0123521 SEQID: 3 claimed sequence
CN 3: PN: WO0123427 SEQID: 3 claimed protein
SQL 14

SEQ 1 AVAEIQLMHA RAKH
========== ===
HITS AT: 1-13

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 135:353089

REFERENCE 2: 134:276166

REFERENCE 3: 134:276165

L4 ANSWER 42 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **294199-44-3** REGISTRY
CN L-Tyrosinamide, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-alanyl-L-arginyl-L-alanyl-L-lysyl-L-histidyl-L-leucyl-L-asparaginyl-L-seryl-L-methionyl-L-.alpha.-glutamyl-L-arginyl-L-valyl-L-.alpha.-glutamyl-L-tryptophyl-L-leucyl-L-arginyl-L-lysyl-L-lysyl-L-leucyl-L-glutaminyl-L-.alpha.-aspartyl-L-valyl-L-histidyl-L-asparaginyl- (9CI) (CA INDEX NAME)
CI MAN
SQL 34

SEQ 1 AVAEIQLMHA RAKHLNSMER VEWLRKKLQD VHNY
========== ===
HITS AT: 1-13

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 133:247378

L4 ANSWER 43 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **293299-25-9** REGISTRY
CN L-Lysinamide, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-alanyl-L-

arginyl-L-alanyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 28: PN: WO0123521 SEQID: 9 claimed sequence
SQL 13

SEQ 1 AVAEIQLMHA RAK
========== ===
HITS AT: 1-13

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 134:276166

REFERENCE 2: 133:247378

L4 ANSWER 44 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **293299-21-5** REGISTRY
CN L-Tryptophanamide, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-alanyl-L-arginyl-L-alanyl-L-lysyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 26: PN: WO0123521 SEQID: 7 claimed sequence
SQL 14

SEQ 1 AVAEIQLMHA RAKW
========== ===
HITS AT: 1-13

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 135:205634

REFERENCE 2: 134:276166

REFERENCE 3: 133:247378

L4 ANSWER 45 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **293299-20-4** REGISTRY
CN L-Histidinamide, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-glutaminyl-L-arginyl-L-alanyl-L-lysyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 27: PN: WO0123521 SEQID: 8 claimed sequence
SQL 14

SEQ 1 AVAEIQLMHQ RAKH
========== ===
HITS AT: 1-13

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 135:205634

REFERENCE 2: 134:276166

REFERENCE 3: 133:247378

L4 ANSWER 46 OF 54 REGISTRY COPYRIGHT 2003 ACS

RN **293299-19-1** REGISTRY
CN L-Histidinamide, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-alanyl-L-arginyl-L-alanyl-L-lysyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 20: PN: WO0123521 SEQID: 3 claimed sequence
SQL 14

SEQ 1 AVAEIQLMHA RAKH
========== ===
HITS AT: 1-13

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 138:33479

REFERENCE 2: 134:305466

REFERENCE 3: 134:276166

REFERENCE 4: 133:247378

L4 ANSWER 47 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **293299-18-0** REGISTRY
CN L-Histidinamide, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-asparaginyl-L-arginyl-L-alanyl-L-lysyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 25: PN: WO0123521 SEQID: 6 claimed sequence
SQL 14

SEQ 1 AVAEIQLMHN RAKH
========== ===
HITS AT: 1-13

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 134:276166

REFERENCE 2: 133:247378

L4 ANSWER 48 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **293299-16-8** REGISTRY
CN L-Histidinamide, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-alanyl-L-leucyl-L-alanyl-L-lysyl- (9CI) (CA INDEX NAME)
SQL 14

SEQ 1 AVAEIQLMHA LAKH
========== ===
HITS AT: 1-13

REFERENCE 1: 133:247378

L4 ANSWER 49 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **293299-15-7** REGISTRY
CN L-Histidinamide, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-alanyl-L-

arginylglycyl-L-lysyl- (9CI) (CA INDEX NAME)
SQL 14

SEQ 1 AVAEIQLMHA RGKH
========== ===
HITS AT: 1-13

REFERENCE 1: 133:247378

L4 ANSWER 50 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **293299-11-3** REGISTRY
CN L-Histidinamide, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-asparaginyl-L-leucyl-L-alanyl-L-lysyl- (9CI) (CA INDEX NAME)
SQL 14

SEQ 1 AVAEIQLMHN LAKH
========== ===
HITS AT: 1-13

REFERENCE 1: 133:247378

L4 ANSWER 51 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **293299-10-2** REGISTRY
CN L-Histidinamide, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-asparaginyl-L-arginylglycyl-L-lysyl- (9CI) (CA INDEX NAME)
SQL 14

SEQ 1 AVAEIQLMHN RGKH
========== ===
HITS AT: 1-13

REFERENCE 1: 133:247378

L4 ANSWER 52 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **293299-09-9** REGISTRY
CN L-Histidinamide, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-alanyl-L-leucylglycyl-L-lysyl- (9CI) (CA INDEX NAME)
SQL 14

SEQ 1 AVAEIQLMHA LGKH
========== ===
HITS AT: 1-13

REFERENCE 1: 133:247378

L4 ANSWER 53 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **293299-05-5** REGISTRY
CN L-Histidinamide, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-asparaginyl-L-leucylglycyl-L-lysyl- (9CI) (CA INDEX NAME)
SQL 14

SEQ 1 AVAEIQLMHN LGKH
========== ===
HITS AT: 1-13

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 133:247378

L4 ANSWER 54 OF 54 REGISTRY COPYRIGHT 2003 ACS
RN **229616-37-9** REGISTRY
CN L-Histidine, L-alanyl-L-valyl-L-alanyl-L-.alpha.-glutamyl-L-isoleucyl-L-glutaminyl-L-leucyl-L-methionyl-L-histidyl-L-asparaginyl-L-leucylglycyl-L-lysyl- (9CI) (CA INDEX NAME)
SQL 14

SEQ 1 AVAEIQLMHN LGKH
========== ===
HITS AT: 1-13

RELATED SEQUENCES AVAILABLE WITH SEQLINK

REFERENCE 1: 131:83087

FILE 'HOME' ENTERED AT 15:49:52 ON 03 JUN 2003